WHAT IS CLAIMED IS:

1. A method of modulating movement of a cell within or to the skin of a mammal, said method comprising administering to said mammal an effective amount of:

a) an antagonist of CTACK;

b) an agonist of CTACK;

c) an antagonist of Vic; or

d) an agonist of Vic.

2. The method of Claim 1, wherein said modulating is blocking and said administering is an antagonist of CTACK or Vic.

3. The method of Claim 2, wherein:

a) said movement is:

i) within said skin;

ii) chemotactic; or

iii) chemokinetic;

b) said administering is local, systemic, topical, subcutaneous, intradermal, or transdermal;

c) said administering is an antagonist of CTACK or Vic;

d) said cell is:

i) a CLA+ cell;

ii) a T cell;

iii) a dendritic cell; or

iv) a dendritic cell precursor;

v) a dermal fibroblast cell;

vi) a dermal endothelial cell; or

vii) a melanocyte; or

e) said cell moves into the dermis and/or epidermis layers of said skin.

4. The method of Claim 2, wherein:

a) said antagonist is selected from:

i) a mutein of natural CTACK or Vic;

ii) an antibody which neutralizes CTACK or Vic; or

iii) an antibody which blocks GPR2 ligand binding;

b) said mammal is subject to a transplant or skin graft;

c) said antagonist is administered in combination with an antibiotic, analgesic, immune suppressive therapeutic, anti-inflammatory drug, growth factor, or immune adjuvant.

5. The method of Claim 1, wherein said modulating is attracting and said administering is an agonist of CTACK or Vic.

6. The method of Claim 5, wherein:

a) said movement is:

i) within said skin;

ii) chemotactic; or

iii) chemokinetic;

b) said administering is local, topical, subcutaneous, intradermal, or transdermal;

c) said administering is a CTACK or Vic ligand;

d) said cell is:

i) a CLA+ cell;

ii) a T cell;

iii) a dendritic cell; or

iv) a dendritic cell precursor;

v) a dermal fibroblast cell;

vi) a dermal endothelial cell; or

vii) a melanocyte; or

e) said cell moves into the dermis and/or epidermis layers of said skin.

7. The method of Claim 5, wherein:

a) said agonist is selected from:

i) CTACK or Vic; or

ii) a GPR2 ligand;

b) said mammal is subject to a cutaneous lesion, tumor or viral, microbial, or parasitic infection;

c) said agonist is administered in combination with an antibiotic, analgesic, immune suppressive therapeutic, anti-inflammatory drug, growth factor, or immune adjuvant.

8. The method of Claim 5, wherein the agonist is administered as a cutaneous adjuvant.

9. A method of purifying a population of cells, said method comprising contacting said cells with CTACK or Vic, thereby resulting in the identification of cells expressing a receptor for said CTACK or Vic.

10. The method of Claim 9, wherein said contacting results in specific movement of said cells to a site for purification.

11. The method of Claim 9, wherein said movement is through pores of a membrane.

12. A method of producing a ligand:receptor complex, comprising contacting:

a) a mammalian CTACK with a GPR2 receptor; or

b) a mammalian Vic with a GPR2 receptor;

wherein at least one of said ligand or receptor is recombinant or purified, thereby allowing said complex to form.

13. The method of Claim 12, wherein:

a) said complex results in a Ca++ flux;

b) said GPR2 receptor is on a cell;

c) said complex formation results in a physiological change in the cell expressing said GPR2 receptor;

d) said contacting is in combination with IL-2 and/or interferon-α; or

e) said contacting allows quantitative detection of said ligand.

14. A method of modulating physiology or development of a GPR2 expressing cell comprising contacting said cell to an agonist or antagonist of a mammalian Vic or CTACK, wherein one of said GPR2 receptor or said agonist or antagonist is recombinant or purified.

15. The method of Claim 14, wherein:

A) said antagonist is:

1) an antibody which:

a) neutralizes said mammalian Vic;

b) neutralizes said mammalian CTACK; or

c) blocks ligand binding by GPR2; or

2) a mutein of said Vic or CTACK; or

B) said physiology is selected from:

1) a cellular calcium flux;

2) a chemoattractant response;

3) a cellular morphology modification response;

4) phosphoinositide lipid turnover; or

5) an antiviral response.

16. The method of Claim 15, wherein:

a) said antagonist is an antibody and said physiology is a chemoattractant response; or

b) said modulating is blocking, and said physiology is an inflammatory response.

17. A method of testing a compound for ability to affect GPR2 receptor-ligand interaction, said method comprising comparing the interaction of GPR2 with Vic or CTACK in the presence and absence of said compound.

18. The method of Claim 17, wherein said compound is an antibody against GPR2, Vic, or CTACK.

19. A primate GPR2, comprising sequence of MGTEVLEQ (see SEQ ID NO: 2).

20. A nucleic acid encoding said GPR2 of Claim 19.

21. An antibody which binds selectively to MGTEVLEQ (see SEQ ID NO: 2).

22. A method of treating a patient suffering from a skin disorder comprising administering an effective amount of an antagonist against GPR2, Vic, or CTACK.

23. The method of Claim 22, wherein the antagonist is an antibody.